Botulism

Bioterrorism Agent Profiles for Health Care Workers

Causative Agent: The botulinum toxins are a group of seven related toxins produced by the bacillus, *Clostridium botulinum*. The seven distinct toxins are produced by different strains of the bacillus.

Routes of Exposure: Inhalation, Oral

Infective Dose & Infectivity: .001 μg/kg (for type A toxin)

Incubation Period: Neurologic symptoms of foodborne botulism generally begin 12-36 hours after ingestion. Neurologic symptoms of inhalational botulism generally begin 24-72 hours after aerosol exposure. However, the incubation period for both can range from 6 hours to 10 days.

Clinical Effects: Botulinum toxins are neurotoxins that act to prevent the release of acetylcholine presynaptically and thus block neurotransmission. Multiple cranial nerve palsies are often the first symptoms seen. Bulbar palsies are prominent early, with eye symptoms such as blurred vision due to mydriasis, diplopia, ptosis and photophobia, in addition to other bulbar signs such as dysarthria, dysphonia, and dysphagia. Skeletal muscle paralysis follows with a symmetrical descending and progressive weakness, which may culminate abruptly in respiratory failure. Deep tendon reflexes may be present or absent.

Lethality: The mortality rate from botulism is 60% if the patient goes untreated and less than 5% if the patient receives appropriate treatment. All the botulinum toxins are slightly less toxic when exposure is by the pulmonary route: a recent estimate of the human LD_{50} by inhalation is 3 ng/kg.

Transmissibility: Botulinum toxin cannot be transmitted person-to-person.

Primary contaminations & Methods of Dissemination: Likely methods of dissemination would be sabotage of food/water supply or aerosol

Secondary Contamination & Persistence of organism: The toxin is not dermally active. *C. botulinum* spores can persist in the environment, and wound botulism can result when an open wound is contaminated by ground-in soil or gravel.

Decontamination & Isolation:

Patients- Patients can be managed using standard precautions. No decontamination is necessary following foodborne exposure. Following aerosol exposure to botulinum toxin, skin should be rinsed with soap and water.

Equipment, clothing & other objects- Hypochlorite (0.5% for 10- 15 minutes) and/or soap and water should be used for environmental decontamination. Clothing should be washed with soap and water. The natural decay rate of the toxin is 1% per minute. Therefore, after 48 hours, there will be 13 logs of decay.

Identification: The best diagnostic sample for immunologic identification of the toxin is from swabs taken from the nasal mucosa within 24 hours after inhalational exposure. No antibody response is induced after exposure due to the small quantity of toxin protein needed to kill. A confirmatory diagnosis comes from mouse bioassays demonstrating toxin in the blood or stool,

neutralized by the appropriate antisera. Many times the organism can be isolated from the offending food, and toxin and neutralizing tests can then be run again using food samples. Diagnostic services are available only through the county and state health departments.

Treatment: Treatment is the same for inhalation (aerosolized) exposure or ingestion (foodborne). Care is supportive. Long term mechanical ventilation may be needed for several weeks to months. A trivalent equine antitoxin for food- borne botulism is available from the CDC through the Arizona Department of Health Services. Use of the antitoxin requires skin testing for horse serum sensitivity prior to administration. Providers should refer to the information sheet that comes with the antitoxin.

Prophylaxis: Currently, there is no commercially available vaccine. Contact the Arizona Department of Health Services for possible investigational drugs.

Differential Diagnosis: Single cases may be confused with various neuromuscular disorders such as atypical Guillain-Barre syndrome, myasthenia gravis, or tick paralysis. Botulism could also be confused with enteroviral infections, but in these patients, fever is present, paralysis is often asymmetrical, and the CSF is abnormal. It may be necessary to distinguish nerve agent and atropine poisoning from botulinum intoxication. In organophosphate nerve agent poisoning pupils are miotic and copious secretions are present. In atropine poisoning, the pupils are dilated and mucous membranes are dry, but central nervous system excitation with hallucinations and delirium is present.

References:

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